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Pathobiology of Simian T-Lymphotropic Virus type III

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It has recently been recognized that the T-Lymphotropic retrovirus family also includes closely related agents that infect certain non-human primate species. Similarities to the human viruses include T4 tropism, *in vitro* growth characteristics, cross-reactive viral proteins of similar sizes and association with similar deseases. Therefore these viruses have been designated as the simian T-lymphotropic viruses (STLV). STLV-III viruses have been described in both captive ill rhesus macaques and healthy wild-caught African green monkeys. The major STLV-III proteins have been identifed by RIP-SDS/PAGE and Western blot as gp120/160, gp32, p64, p55, p53, p24 and p15 similar to and cross-reactive with the major *env, gag,* and *pol* encoded products of HTLV-III/LAV. As in the case of HTLV-III infected people, the gp120/160 appears to be the best serologic marker for infection by these closely related viruses by RIP-SDS/PAGE analysis. Serologic studies on a variety of African primates indicated that approximately, 50 % of wild-caught African green monkeys (*Cercopithecus sp*) were seropositive for STLV-III AGM, whereas chimpanzees, baboons, patas monkeys and colobus monkeys were seronegative. STLV-III_{AGM} antibody positive serum samples from *Cercopithecus sp* have been identified from animals sampled as early as 1961. African green monkeys were reportedly transplanted to the Caribbean over 200 years ago. Notably, 0 of 98 (0 %) of these primates were seropositive for STLV-III_{AGM}.

Studies with STLV-III_{mac} have indicated that this virus in the macaque host is closely linked to an immunodeficiency disease, similar to human AIDS. To date there has been no evidence of disease in any African green monkeys that have evidence of exposure to STLV-III_{AGM} in over 600 samples tested. Understanding the biology of an HLTV-III-related virus in this primate species may help us understand the specific viral alterations or viral-host interactions that are involved in the pathogenicity of this family of T-lymphotropic retroviruses and perhaps provide a new approach in the development of an AIDS vaccine.

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Identification of new human T-Lymphotropic retrovirus related to STLV-III of African green monkeys (STLV-III^{AGM})

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AIDS cases are believed to have been present in Central africa in the mid-1970's prior to the recognition of the disease in the United States and Europe. More recent studies in Central Africa indicate that AIDS is endemic in this locale with significant transmission occuring wthin the heterosexual population. Serologic studies of HTLV-III/LAV, the etiologic agent of AIDS, indicate that this or a related virus may have been present in Africa for over a decade. The close relationship of STLV-III to HTLV-III prompted us to investigate further the possibility that these viruses may have shared a common origin. We speculated that STLV-III may have been transmitted to man, at some time during the natural history of these viruses. We hypothesized that there might exist variant strains of HTLV-III/LAV that would be detectable using appropriate serologic probes. STLV-III_{AGM} provides such a probe since it has been demonstrated that this simian member of the HTLV-III/LAV family is related to the human virus by major antigen cross-reactivity, bidirectionally across species lines.

In contrast to previously studies human serum samples, serum samples from apparently healthy West African people demonstrated unique serologic profiles to HTLV-III and STLV-III antigens. These sera demonstrated strong reactivity to all of the major viral antigens of STLV-III_{AGM} with absent or variable reactivity to the major viral antigens of HTLV-III by RIP-SDS/PAGE and Western blot analysis. This suggests that this new human virus shares more common epitopes with SLTV-III_{AGM} than with the prototype HLTV-III/LAV that infects people in the United States and Europe. Further study on the origin of the HTLV-III/LAV group of viruses such as the agent present in West Africa may expand our understanding of the unique pathogenicity of the human AIDS virus.

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The family of T-Lymphotropic viruses in primates and humans

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At present, HTLV-III/LAV, the causative agent of AIDS, has the coding capacity for at least 6 proteins. Similar to other retroviruses, there are p55, p24, p17, and p15 *gag* encoded proteins. We first described the *env* proteins of HTLV-III/LAV as a precursor gp160 and external gp120 — these being the most immunogenic proteins in people exposed to this virus using radioimmunoprecipitation. Studies in our lab as well as others have shown that the p27, *3'orf* product, p23 *sor* product, p53, p64 *RT* proteins, and p34 *endonuclease* proteins are immunogenic in certain individuals exposed to this virus. We have therefore speculated that these serologic markers may provide useful information of prognostic value.

It has been recently recognized that the T-lymphotropic virus family also includes closely related agents that infect certain primate species. STLV-III viruses have been described in both captive ill rhesus monkeys and also in a large number of healthy African green monkeys. STLV-III demonstrates T4 tropism, *in vitro* growth characteristics and ultrastructural morphology similar to HTLV-III/LAV. The major STLV-III viral proteins are all similar in size and serologically cross-reactive with the major viral proteins of HTLV-III.

The close relationship of STLV-III_{AGM} to HTLV-III raised the possibility that the simian virus may have been transmitted to humans at some time during its evolution. It is therefore possible that a range of viruses exists, perhaps with differing pathogenicity and relatedness to STLV-III_{AGM}. We recently isolated a new human virus designated HTLV-IV, from apparently healthy people in West Africa. The present data suggest that HTLV-IV shares more common epitopes with STLV-III than with the prototype AIDS virus. Further study of HTLV-IV may contribute to understanding of how the HTLV-III group of viruses originated and how their unique pathogenicity can be prevented.

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